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ULCER OF THE STOMACH AND DUODENUM*

E. STARR JUDD, M. D.,
Rochester, Minnesota

Gastric ulcer was recognized and reported as early as 1600. Travers, in 1817, described two cases of perforating duodenal ulcer. But apparently all of the factors which enter into the production of ulcers in the gastro-intestinal tract are not yet known, in spite of the great amount of experimental work and detailed studies that have been carried out in the past century and a half. In some instances it is impossible to demonstrate any condition which might even be a remote factor in the etiology of the lesion. That many benign ulcers fail to produce symptoms and heal spontaneously, while for some unknown reason others refuse to respond to any kind of treatment, is further proof of the breadth of the problem that still confronts us. So far as I know, nothing of great significance has been learned recently regarding the cause of ulcer. It may be that the changing conditions under which we live are responsible for the increasing predominance of certain types of lesions and the consequent variation in the symptom complex. Undoubtedly this adds to the complexity of the problem, but on the other hand, closer observation of the trend of events may lead the way to some newer and better approach to its solution.

It is my impression that in the past year or two more patients have presented themselves with bleeding duodenal ulcer than ever before. In many of these, hemorrhage had occurred suddenly and without the usual precedent of dyspepsia or other symptoms of ulcer. Although the history given was that of severe bleeding, nevertheless, at operation it was often impossible to demonstrate the presence of a definite ulcer as the point of origin of the hemorrhage. Instead of this, through much of the first portion of the duodenum there was an extensive inflammatory condition which we designate as duodenitis. We were unable to find any crater type of lesion or any break in the mucous membrane.

Furthermore, it is a matter of much interest that physicians returning from European clinics report that they have observed a marked dissimilarity between the lesions seen there and those which they see among their own patients in this country. The condition which we usually find is a comparatively small localized area of inflammation in the cap of the duodenum. Not infrequently, on opening the intestine, we discover a second but smaller lesion on the posterior surface of the duodenum. Sometimes there is a little crater ulcer associated with the area of inflammation, but this is not always present. The lesions seen in patients observed in European clinics are reported as being much more extensive and severe. This undoubtedly accounts for the fact that the radical operation for duodenal ulcer is advocated so widely, particularly in Germany and Austria. Surgeons on the Continent feel that removal of as much as possible of the lesser curvature of the stomach with its acid-bearing glands reduces the incidence of secondary ulcer.

The ulcers which we find in the stomach are likely to be discrete lesions; sometimes there is considerable associated gastritis but usually the remaining tissue of the gastric wall is not greatly changed. In striking contrast to this, European surgeons are reported as being called on to treat many cases in which there is extensive gastritis, usually with one or more associated ulcers. It has been said that these patients do not respond to alkalinization, rest, and other methods of treatment, that they cannot be cured by any type of conservative operation, and that the only plan offering permanent relief is radical operation, which removes all of the greatly thickened and inflamed gastric wall. Aschoff's studies of material obtained at necropsy of soldiers killed in battle revealed that extensive and more or less general gastritis was rather prevalent. This led him to suggest that the condition might be the cause of ulcer.

* Read before the Medical Society of Delaware, Wilmington, October 14, 1931.

If we were dealing with the same type of lesion as these European surgeons, I think that we would probably feel as they do about the method of treatment.

PATHOGENESIS

In any study of the pathogenesis of ulcer, one must take many factors into consideration. First of all, much confusion arises from the use of the term "peptic ulcer." It should be recognized that ulcers of the stomach and duodenum are separate and distinct lesions. Second, the lesions are peculiar to man, which makes experimental study difficult. A review of some of the plans that have been advanced for the investigation of this subject will not be amiss, for they show the numerous angles from which the problem has been approached and the wide variation in the deductions that have resulted.

Cohnheim simplified the problem somewhat by dividing it into two parts: the cause of the original defect, and the cause of the chronicity. Certain observers have expressed the belief that ulcers are the issue of the combined action of localized vascular disturbance with focal nutritional changes and the digestive action of the gastric secretion on the devitalized tissue.

The characteristic ulcer of human beings is situated on the lesser curvature of the stomach, in the pyloric mucosa, or just distal to the pylorus in the duodenal mucosa. It thus occurs in that region of the gastro-intestinal tract which may be exposed to an acid medium and on which the gastric content may impinge with considerable force during the passage of food from the stomach. These two considerations form the basis of Mann's investigations. Research workers found that it was difficult or impossible constantly to expose the gastric mucosa to acid medium by administering acid. It was thought that this might be brought about by eliminating the secretions poured into the duodenum beyond the pylorus, which tend to neutralize the acid that had not been neutralized in the stomach. This has been accomplished by various types of operations on experimental animals, but the most satisfactory method for producing subacute or chronic ulcer is to perform duodenal drainage as is done by Mann. The procedure causes the gastric content to be expelled from the stomach into the jejunum without being mixed with secretions poured into the duodenum. An ulcer develops in almost every animal in which the procedure is carried out. The ulcer is always of the crater type and it is almost identical with that seen in the stomach or duodenum of human beings.

In carrying out experimental researches on the liver, Bollman and Mann found that duodenal ulcer forms following partial hepatectomy or establishment of Eck fistula, and in some instances after ligation of the common bile duct. The lesion occurs with sufficient frequency to interfere with experiments on the liver. In many instances the ulcer perforates and the animal dies. Of course, it is possible that trauma is a factor in the production of some of these lesions. Whether chronic ulcer of the duodenum may be brought about by some disturbance of function of the liver is an interesting speculation. That an acute ulcer may become chronic is a reasonable assumption. Nevertheless, it is an open question whether the occurrence of chronicity involves fundamental principles other than those which are concerned in the origin of the original defect.

Disturbance of the vegetative system has also been advanced as a cause of ulcer. There are many clinical facts which suggest that this neurogenic theory should be given consideration. Our experience during the past few years shows that we are able completely to control the symptoms of ulcer by the use of sedatives such as luminal. Patients with very high values for gastric acids, and with severe symptoms, have been entirely relieved by the use of one or two doses of sedative; in some instances the acidity returned to normal. It is not my intention to suggest continuous treatment of this kind, for such a plan must not be entertained. According to the neurogenic theory, nervous factors produce spasms either of the muscles or of the vessels in localized areas and cause the mucosal defect. From the results obtained with sedatives, it seems that disturbance of the nervous system must be a considerable factor in certain cases.

Konjetzny advanced the theory of the inflammatory origin of ulcer. In his gross and microscopic study of tissue removed at operation, he observed that all of the stages from simple erosion to chronic ulceration may be found in a single resected specimen. It is this type of lesion that is so prevalent in Germany and Austria at present.

Many experiments have been carried out to determine the rôle of bacteria in the formation of ulcer. Strains of streptococci recovered from tonsils and infected teeth of patients with ulcer or inflammation in the duodenum, when injected into animals, produce lesions closely resembling those seen in human beings. Although an ulcer strain of organism frequently has been obtained, chronic ulceration in the stomach or duodenum practically never has been reproduced. In a large proportion of the animals there will be what might be termed acute or subacute ulceration in the stomach or duodenum, but these lesions usually heal promptly. I do not believe this means that we should disregard foci of infection. While it is difficult to say whether cure is obtained by removal of the foci, nevertheless, the evidence is too great to be disregarded. Infection in teeth, tonsils, sinuses, prostate gland, and so forth should be eradicated regardless of whether the patient undergoes surgical intervention for the ulcer.

DUODENITIS

My daily observations force me to conclude that the lesion found in the duodenum is more often duodenitis than duodenal ulcer. The inflammatory area is usually small and situated close to the upper edge of the duodenum, about 2.5 cm. or less below the ring of the pyloric muscle. The inflammation may extend to the upper edge of the duodenum; not infrequently it passes around the wall to the posterior aspect, and in some instances the entire circumference is involved. Although the condition is spoken of as ulcer, frequently there is no perceptible break in the mucosa even though profuse bleeding has occurred. Microscopic sections of this same tissue will show the usual evidence of inflammation with much round cell infiltration and edema. Often the duodenal wall is not thickened; in fact, it may seem thinner than the normal intestinal wall. Time and experience with this condition have proved that such a localized area of inflammation is not just a stage of what is later to become a true crater lesion, but that it may continue indefinitely without visible ulceration. On the other hand, microscopic study may reveal the presence of ulcers in these areas of inflammation. And frequently there is considerable duodenitis associated with an ulcer.

CLINICAL FEATURES

The diagnosis of duodenal ulcer is usually readily made from the clinical history and then confirmed by roentgen examination. One of the interesting features is the seasonal nature of the complaint. It is difficult to understand how a patient will have all of the usual symptoms during the spring and autumn, which are not easily controlled with most rigid treatment, and then within a few days, have complete freedom from symptoms. During this period of freedom he may be quite indiscreet about his living and his diet, and still not have any return of symptoms. Looking at this from the standpoint of the lesion itself, it certainly is not reasonable to assume that the ulcer healed during the period of remission of symptoms. It also seems difficult to explain this feature of the complaint on the basis of disturbance in the nervous system.

As I have said, it seems to me that more patients with bleeding ulcer have been seen during the last few years than formerly. Bleeding from these lesions is often severe, and may place the patient in a serious condition, although loss of life from such bleeding is very unusual.

Rivers and Wilbur have recently completed a study of the diagnostic significance of hematemesis. They reviewed 668 histories of cases. In 235 of the cases, operation had not been performed, but the diagnosis had been made after careful clinical investigation. In 432 of the 668 cases, surgical exploration was carried out. They noted that duodenal ulcer alone was responsible for hemorrhage in half of the total number in this series of cases, and gastric ulcer was responsible for only 6.4 per cent. Carcinoma was the cause in less than 13 per cent. They expressed the belief that the source of the hematemesis usually can be determined with accuracy if data obtainable through a detailed anamnesis, careful general examination, and laboratory studies are evaluated cautiously. They also said that indigestion and hemorrhage mean usually an intrinsic gastro-duodenal lesion.

These duodenal lesions occur more frequently in males than in females. The age incidence is difficult to determine, for the prevalent periods of remission of symptoms help to make the history obscure and encourage delays in seeking treatment. In other cases, rapid onset and severity of symptoms have caused the patient to seek attention early in the course of the disease. Some were in their teens, but the majority were in the middle span of life.

TREATMENT

In the presence of conflicting views regarding the origin of ulcer, different forms of treatment are justified only on the basis of experience. A sharp line must be drawn between gastric and duodenal ulcer. In the first place, in considering the ulcer problem it is necessary to keep in mind the high incidence of malignancy in the gastric lesions and the predominantly benign nature of those occurring in the duodenum, for barring other considerations such as hemorrhage, the treatment of these conditions should be tempered accordingly. No matter how small a percentage of gastric ulcers is malignant or will become malignant, the possibility that they will become malignant at all adds to the seriousness of the disease. Radical operation is preferable for gastric ulcer, if there is considerable associated gastritis.

Many duodenal ulcers heal permanently under medical treatment. Anyone with a duodenal ulcer is entitled to a trial of this type of treatment, and to continue with the regimen as long as it is helpful. It is not a good plan to operate when the lesion is bleeding. If the patient has had one hemorrhage only, and is free from symptoms at the time of the examination, it is not necessary or advisable to operate immediately. If he has a recurrence of symptoms, then an operation may be indicated. Should surgical treatment become necessary, particularly because of repeated severe hemorrhages, it is imperative to remove the ulcer if it can be done thoroughly by excision, taking part of the muscle of the pyloric sphincter. With this removal of muscle, everything is accomplished that can be expected of gastro-enterostomy, and, in addition, the ulcer is removed. The operation of excision was developed to be used in those cases in which it was possible to carry it out safely, with the idea of avoiding jejunal ulcer and possibly reducing the number of cases in which bleeding continues after gastro-enterostomy for hemorrhagic ulcer. This operation can be performed in about 50 per cent of the cases of duodenal ulcer, and in these, better immediate and ultimate results will follow than from gastro-enterostomy. A study of the files shows that local operation can be done with very little risk. In 1,363 cases there was a mortality of 0.44 per cent. The procedure is limited to those cases in which the duodenum is fairly mobile. As one's experience with these cases increases, however, one realizes that it is not difficult to mobilize a duodenum that is fairly firmly fixed, and this should be done in cases in which excision of the ulcer is definitely indicated. Should this not be feasible because of the local condition, then it is advisable to resect the stomach with the ulcer in cases with severe repeated hemorrhages.

The local operation of excision of ulcer, with the cap of the duodenum and a considerable part of the pyloric sphincter completing the operation as a gastro-duodenostomy is the operation of choice in all cases of ulcer of the duodenum, or duodenitis, and in ulcers on the gastric side near the pylorus. In cases in which multiple ulcers are encountered and in which it is not possible to remove all of them, it is probably best to remove the anterior ulcer, close the opening in the duodenum, and then to complete the operation with gastro-enterostomy. In patients past middle age with obstruction of the duodeum, gastro-enterostomy gives satisfactory results. There is sure to be complete relief of symptoms, with little probability of the formation of secondary ulcers. It is satisfactory in all cases except in those in which secondary ulcers do develop, and in those in which hemorrhage occurs and in which bleeding may continue.

So far as our present knowledge is concerned a person who has once had an ulcer must always be considered as thenceforward having an ulcer potentiality. Although it is not necessary for him to adhere rigidly to dietary restrictions, nevertheless at any suggestion of a return of his former symptoms, he should be very cautious about his diet and activities. If he is willing to co-operate in this plan of treatment, there is no reason why he cannot be made comfortable and live a very useful and active life; if he does not follow the regimen, and symptoms recur, they are likely to be much more difficult to control than the original disturbance.

When the problem of the etiology of ulcer of the stomach and duodenum and duodenitis is solved, possibly we shall be obliged to change the methods of treatment in some respects. We have already made great progress in the control of these conditions, and in a large proportion of

the cases we are able to obtain relief from the symptoms. However, we shall not be able to assure every patient of a complete and permanent cure until we know the exact cause of the lesions.

DISCUSSION

DR. W. WAYNE BABCOCK (Philadelphia): I should like to at least say a word of appreciation of this paper. Dr. Judd with his enormous experience and skill has clarified the subject, as he always does, and has brought to us ideas of which we may have had fragmentary knowledge, but regarding which we have not as complete knowledge as we should like; for example, his statement that after an ulcer of the stomach one need not fear an ulcer from gastrojejunostomy. That gives one a great deal of comfort in doing his operation; and his reference, which to me was entirely new, to the more serious lesions found abroad as having a bearing on this great wave of very drastic resections there being done was very enlightening and explanatory.

I wish simply to add a word of appreciation.

DR. GEORGE W. VAUGHAN (Wilmington): I should like to ask Dr. Judd as to the percentage of these cases of pure duodenitis that proved to have any gall tract infection, as demonstrated either by xray study or by duodenal tube, and as to their findings, especially the microscopic findings of the duodenal fluid in these cases.

DR. E. STARR JUDD: I want to thank you again for the opportunity of being here. I will try to answer that question. I do not think there is any difference in those cases, as far as the contents of the duodenum are concerned than the ordinary ulcer tract. The association between biliary tract disease and ulcer is a very common thing, but the exact proportion I am not ready to state.

American Board for Ophthalmic Examinations

The American Board for Ophthalmic Examinations will hold an examination in New Orleans on Monday, May 9th, 1932, at the time of the meeting of the American Medical Association.

Necessary applications for this examination can be procured from the Secretary, Dr. William H. Wilder, 122 South Michigan Avenue, and should be sent to him at least sixty days before the date of the examination.

KINETOCYTES* A NEW (Fourth) BLOOD ELEMENT

Preliminary Report

HYMAN I. GOLDSTEIN, M. D.
Camden, N. J.
LEOPOLD GOLDSTEIN, M. D.
Philadelphia, Pa.
and
HENRY Z. GOLDSTEIN, M. D.
Newark, N. J.

EDITOR'S NOTE: Dr. H. I. Goldstein first began the study of this new blood element with Primarius Dr. Adolf Edelmann, Director of the S. Canning Childs' Hospital and Research Institute (Vienna), while spending some time in Carlsbad, Czecho-Slovakia, and in Vienna (1931). On one occasion Primarius Edelmann and Doctor Goldstein studied blood preparations taken from the latter (H. I. G.) after twenty-seven hours absolute fast (no food and no water whatever). Several actively motile "Kinetocytes" were found in Doctor Goldstein's blood after this complete starvation test—in the preparations examined by Edelmann and Goldstein, and made by the laboratory expert, Loewi, of Carlsbad.

Since Swammerdam (1658), van Leeuwenhoek (1673) and Kircher (1673), Muys, Della Torre, von Stahl, Boerhaave, von Haller, Hunter, Hewson, Prevost, von Gruithuisen, and Edwards (1821) made known the results of their studies on the blood many others have made valuable contributions to our knowledge of this subject. We need mention only a few of the more important: Malpighi (1628-1694), the founder of histology, described the red blood cells in 1665 (seven years after Swammerdam), as "fat globules looking like a rosary of red coral." It was Leeuwenhoek (1632-1723), of Delft, who first described the spermatozoa after Hamen, in 1674, called his attention to them. Leeuwenhoek gave the first complete account of the red blood-corpuscles (1673, 1674). Swammerdam (1637-1680) was perhaps the first to discern and describe the red blood-corpuscles. He also discovered the valves of the lymphatics. Metchnikoff, in 1884, described phagocytosis in leucocytes. Ehrlich (1898) classified the leucocytes. The earliest observers of mammalian blood-platelets were probably Donné, Zimmermann (1846-1848), and Arnold (1845). Donné called them "globulins." Bizzozero (1882) called them "blood-platelets," while Hayem called them "hématoblasts." They are now known, through Dekhuyzen, as "thrombocytes." Wright (1910) believed that the platelet is derived from the megakaryocyte of the bone marrow. "Motile" (Brownian) micro-thrombocytes have been described by Pelczar and Koloszynski (1931) in rats.

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Mention should be made of Nasse (1835), Rouget (1873-1879), Andral (1843), Virchow (1845), Hodgkin and Lister (1827), Burkhart (1828), Krimer (1834), Dollinger, Rudolphi (1771-1832), Burdach, Magendie, Schultz, Wagner, Gerlach (1849), Kolliker, Pappenheim, Rindfleisch, Reitter, Biermer, Naegeli, Turk, Schilling, Morawitz, and others.

For many years much has been written on the three well-known blood elements: erythrocytes, leucocytes, and thrombocytes. Many and varied have been the normal and abnormal findings and changes described in these elements, resulting in the firm establishment of an important branch in medicine—haematology.

In the Wiener klinische Wochenschrift (44: 795 (June 19), 1931), Edelmann published a brief report concerning "A Hitherto Unknown Blood Element." An actively motile, oval or globular body, that can not be confused, after careful study, with the now well-known "fatty hemoconia bodies," described by H. F. Muller (1896). In the near future, perhaps, one will be able more accurately and more definitely to identify this new element, to which Edelmann has given the name *"Kinetocyte,"* and to differentiate it from all other motile and non-motile bodies, pseudospirochetes, pseudoparasites, innenkorper, Heinz-Ehrlich bodies, Turk bodies, etc., that have previously been reported. It is known that a fatty meal does not affect the kinetocytes as the haemakonien (lipoid) bodies. That their presence and activity are not due to warm temperatures or a hot platinum loop (we use no loop). They are best seen in a very thin smear, made from a minute drop of blood, taken in the morning before breakfast. They are small, probably 0.5-1 micron up to 2 microns in size, and are *found moving about in all directions* between the immovable stationary erythrocytes, leucocytes, and thrombocytes. Normally, there are, as a rule, about 1 to 3 in a field. There are, perhaps, 30,000-40,000 in one cubic millimeter of blood. These bodies must not be confused with the "fat globuli" (hematokonien) first described by H. F. Muller, in 1896, and which, according to Neumann, Weltmann, Knudson and Grigg, Gelli, and others, consist of fat.

These kinetocytes will probably, after further investigation, be shown to be different from the motile and non-motile bodies, "microthrombocytes" and "pseudospirochetes," previously reported and described by van Niessen (1895), Beale (1864), Preyer (1864), M. Schultze (1865), Lewaschew (1892), Siegel (1905), Arnold, Eve (1907), Porter (1907 and 1913), Balfour (1911), Seidelin (1912), Eberson (1920), Thoinot and Calmetter (1892), Chambers (1913), E. W. Schultz (March, 1923), Gaule, Zeller (1923), Determann (1925), Demnitz (1926), P. Morawitz (1926), Crawley, Grawitz, V. Schilling (1926, 1929), Grassberger (1928), Zadek and Burg (1930), Rindfleisch (1863), Pelczar and Koloszynski (1931), Meesen, Jungmann, Wlassow, Love, Naegli, Buchanan (1909), Mackay (1931), W. Jones (1846), Schmaus and Albrecht (1895), Breeze (Tangiers) and Foran (Nigeria), and Engel (1893).

They are found in the blood of healthy individuals. They may vary in size, form, activity, and numbers in certain pathological states. Thus, Edelmann found them greatly increased in a young female patient suffering from thrombopenic purpura (*kinetocytosis* or *kinetocythemia*). In several cases of malignancy they appeared to be diminished (*kinetocytopenia*). According to Edelmann they may participate in some way in the blood-clotting process ("Gerinnungs Prozess"), perhaps they possess an antithrombin-like action. Further studies must be made.

We are well aware of the thirty-five or forty reports in the literature, after a careful study of the available literature for the past sixty-five or seventy years, concerning variously described "bodies," "motile cells," "korper," "micro-thrombozyten," "pseudospirochetes," "pseudoparasites," etc.

We publish this as a preliminary report on "kinetocytes," probably a new (fourth) blood element. Perhaps, in the not too distant future, one will be able, definitely, to identify and to differentiate these kinetocytes from all other previously described "bodies," with which this new element may be confused. Investigations should be made as to cultural, staining, temperature, and chemical reaction and response, and as to variations in form, size, number, and motility, etc., in blood dyscrasias, hemorrhagic diatheses, infectious diseases, anemias, leukemias

(leucosis), polycythemia vera (primary and secondary), erythrosis or erythrocytosis, reticulo-endotheliosis, malignancy, athrombopenic and thrombopenic purpura, aleukemic (myeloid) leukemia, (monocytic) histiocytic leukemia, primary hypochromic anemia or idiopathic hypochromemia—cryptogenic achylic chloranemia or achylic chloranemia and megaloblastic hyperchromic anemia (primary pernicious anemia), Goldstein's Disease or heredofamilial angiomatosis with recurring familial hemorrhages, hemophilia, hemolytic jaundice, coronary thrombosis, asthma, diabetes, myeloblastic leukemia, and in splenectomized patients, etc., and studies should be made in normal adults, children, and infants.

Max Schultze (1865) speaks of certain small non-motile bodies in the blood. He refers to the studies by von Rollet, Wharton Jones (1846), and Rindfleisch (1863). Edwin W. Schultz (1923), of Stanford University, discussed pseudospirochetes derived from red blood cells. These pseudospirochetes and other products of corpuscular disintegration follow the application of a hot platinum loop to a drop of blood. They are not "ameboid."

Buchanan (1909) thought haemoconia consist of fibrin. Neumann and others showed that they are really fat particles possessing Brownian movement. They are more numerous after a meal and disappear if the person or animal is starved. They may be absent in persons suffering from disorders of the alimentary tract.

Crawley also studied these small "bodies" and some apparently intra-corpuscular particles (motile) and "beaded threads."

Balfour (October 1, 1909), studied the "bodies" found in the fresh blood of fowl. He reviews the literature, thoroughly, in his paper on "Fallacies and Puzzles in Blood Examination" (Fourth Report of The Wellcome Tropical Research Laboratories, Khartoum, 1911, 109-125).

Walther Kruse (1890) in his paper "Ueber Blutparasiten" mentions the peculiar "bodies" (Korpern) found by O. Butschli and Gaule in frogs' blood. Kruse speaks of the small actively motile "Wurmchen und Korpern" in the blood.

Helen Chambers (1913) and Porter (1913) reported "A New Spirochaeta Found in the Human Blood."

Grassberger (1928) found his pseudospirochetes especially increased in icteric patients. These were not actively motile. They came from red blood cells, and they were not thrombocytes, and were found in pernicious anemia and in normal human blood.

Love found haemoconia especially numerous in the blood of typhus patients.

Eberson, of St. Louis (1920), also reported on "spirochetes" derived from red blood-corpuscles. His studies were made in experimental syphilis in the rabbit. These "bodies" were seen in normal and spirochete-infected testicles.

Beale (1863), in his observations upon the nature of the red blood-corpuscle, calls attention to "fine threads" exhibiting vibratory movements, oscillating in many different places at the same time. Many exhibit a distinctly beaded appearance; and when these thin filaments are detached they certainly very closely resemble bacteria. He says, "We must not mistake such particles for bacteria. The movements are molecular in their nature, and are not dependent upon any vital properties. They continue until the fluid becomes so concentrated by evaporation that no further movements in the particles can occur."

Wlassow (1894), of Moscow, in his paper, discussed the blood platelets and emphasized the important part played by them in the blood-clotting process. He speaks of "korperchen" breaking off from the red blood cells and refers also to M. Schultze's "Degeneration Forms."

The majority of the motile bodies (pseudospirochetes) studied by Edwin Schultz were 0.5 micron in thickness, and the entire thickness of the body is illuminated when viewed with the darkfield. He mentions globoid bodies, with flagellae, and various bizarre bodies. He believes heat, fevers, etc., tend to increase their number and forms. They are not corkscrew spiral in shape, never taper to a point, they never exhibit rotary motion around their long axis, with forward and then backward movements. He states "in 1919 he examined about fifty specimens of blood from cases of typhus fever with the darkfield. These pseudospirochetes were present in large numbers in practically every specimen examined."

Determann, of Dusseldorf (1925), and Meessen (1925) found spirochetes (spirochaeta corynephora) in the blood of pernicious anemia pa-

tients. Determann found them also in normal individuals, and in patients suffering from tuberculosis, cancer, etc. These spirochete-like bodies showed no similarity to Siegel's (1905) "cytorhyktes luis" or Doehleschen "korperchen" found in scarlet fever.

Turk described "haemamebae" in myeloblastic leukemia. These were probably artificial products. Turk (1904) thought that the "haemaconia" were "squeezed-out" leucocytic granules, while Grawitz (1911) considered them as lipoid particles derived from the red blood-corpuscles.

Lowit (1899, 1900), Vittadini (1900), and Edelmann (1931), and others, wrote on the "parasitic" etiology of myeloid leukemia.

Turk concludes that Lowit's "hamameben" are artificial products. He says "Sie bleibt bestehen, und die Haemamoeba leucademiae magna ist und bleibt demnach ein Kunstproduct." (E. Ziegler's Beitrage zue pathologischen Anatomie und zur allgemeinen Pathologie (Jena) 30, 371-412, 1901).

Jungmann and Kuczynski (1917) describe motile "diplobazillen—ahnliche Gebilde, die bald als Achter,—bald als Hantelformen imponierten." These were similar to the forms found by His. Topfer (1916) saw similar "bodies" in "His-Wernersche Krankheit" or "Wolhynica Fever." These bodies "Vor und wahrend des Anfalles findet man im steril entnommenen frischen Blutpraparat in der Mitte schwacher, an den Enden starker lichtbrechende Stabchen, die hantelformig aussehen und eine lebhaft taumelnde Fortbewegung haben. Ihre Lange betragt etwa den vierten Teil eines Erythrozytendurchmessers." Jungmann and Kuczynski state that these have nothing to do with the so-called "pseudospirochaten," "deren Abschnurung von roten Blutkorperchen wir haufig beobachtet haben. Diese zeigen peitschende Bewegung und rotierendes Weitergleiten im Plasma. Unsere oben beschriebenen Hantelformen finden sich gelegentlich auch im *trocken fixierten* Ausstrich-praparat, doch sind sie hier schwer auffindbar."

Naegeli (1923) discusses the "amoboiden Bewegungen" of the white blood-corpuscles (page 204). He mentions the presence of "feine kornchen mit lebhaften amoboiden Bewegungen. Entivender sind diese Gebilde sinzeln und in der Grosse sehr variabel oder aber in kleine Ketten geordnet. H. F. Muller hat auf diese Hamatokonien oder Bludstaubchen zuerst aufmerksam gemacht." He quotes Neumann that "ein Grosser Teil der Blutstaubchen aus Fett besteht, und dies kann durch die starke Zunahme nach Mahlzeiten bewiesen werden." (Blutkrankeiten und Blutdiagnotsik, Julius Springer, Berlin, 1923, 185).

Zeller (1923) found in examining blood with the darkfield, corpuscular formations in the shape of rods or filaments, which could not be classified as blood platelets or hemokoniae. These formations were observed by several investigators. Some described them as newly discovered inciting agents and some as artificial products—(Grawitz, Dietrich, Werner, Schilling, Bechhold, Oelze). In the literature available to the author, he "never found these formations described as a regular component of the blood."

Zeller states that in the citrated blood of normal individuals, nearly every visual field reveals rods, all of which have the same length and the same appearance. These rods are somewhat smaller than erythrocytes, and are apparently in active motion, moving about continuously. They are thickened in a knob-shaped manner at both ends and may even move under the erythrocytes. They have an edge and are refractile. Even when observed for a long time, they show no granulation. With Giemsa stain they assume the same color as the platelets. An exact count is not possible, since they appear in a much lesser number in the smear specimen than in the darkfield. It is estimated that these rods are decreased in number when the number of platelets is decreased. Especially in acute infectious diseases one must frequently search for a long time to find them.

Zeller found regularly in the citrated blood of febrile patients, especially in typhus fever, very thin, hardly visible, worm-like tortuous fibrils, which were recognizable only in a dull green light. They were of various lengths, up to four times the diameter of the erythrocytes. (These were probably so-called *"pseudospirochetes."*) When sought for a sufficient length of time they will also be found in the healthy blood. They will not show any granulations even after long observation. They are difficult

to stain with Giemsa solution. These filaments are easily detected in smallpox, scarlet fever, typhus, purpura, all of which are diseases which may be accompanied by a hemorrhagic diathesis. They were found increased most in two cases of purpura fulminans, also in one case of lymphatic leukemia. A slight increase occurred in pulmonary tuberculosis, chronic nephritis, especially when uremia is imminent, in psoriasis, fibromatosis and in some cases of chronic anemia. Nothing definite is known regarding the origin of these "Zeller" rods. Their constant form and size speak against the development as a reaction or decomposition product. In the case of the filaments, conditions are different. Their length and thickness are inconstant. The formation of filaments on erythrocytes has already been observed by the abovementioned investigators, who regarded them as fibrinous threads, lipoids or erythrocyte centers. Whosoever has seen the rigid fibrin threads and needles will never confuse the flexible soft filaments which are under discussion here with the former. The filament formation may very easily be demonstrated in hypotonic solutions of citrated blood, besides free floating filaments of various length, which float free with one end. The number of filament-forming erythrocytes varies greatly according to the disease. All in all, it is quite probable that the filaments come from the erythrocytes.

Determann (1925) carried out examinations in eight cases of pernicious anemia similar to Meessen. With darkfield examination, Determann confirmed Meessen's findings. Following several unsuccessful attempts of staining, he succeeded in demonstrating these organisms which Meessen desires to term "spirocheta corynephora."

As was the case with Meessen, Determann was not successful with the usual anilin stains. Even with Burri's method, no perfect results were obtained. On the other hand, staining with a solution of fifteen (15) drops Giemsa solution ("Grubler") in 10 c. c. of water, with addition of 5-10 drops of potassium chlorate solution, proved successful. Staining of thin smears was then carried out in the case of healthy individuals and in patients who positively did not suffer from pernicious anemia. The same formations were found by Determann in six of ten healthy individuals, and in fifty-eight of eighty-four patients who did not suffer from pernicious anemia.

The fact that these formations were also found by Determann in the majority of healthy individuals and of patients who did not suffer from pernicious anemia proves that they have no specific-etiological significance. A film which was prepared by the Delca-Bioskop Limited constitutes a further proof that these formations also occur in the blood of normal individuals because they were distinctly visible in darkfield kinetoscopic exposures.

Determann raises the question whether these spirochete-like formations have any significance. He considered them somewhat similar to the "cytorhyktes luis" (Siegel) and to Doehle's bodies, which he regards as the etiologic agents of scarlet fever. If it is assumed that these formations are blood-dust, then it is obvious that they will more readily be found in pernicious anemia than in healthy individuals and in patients who do not suffer from a disease causing blood destruction. One must conclude that there is no evidence for the assumption that these protozoa formations or "spirochetes" are responsible for the development of pernicious anemia.

Demnitz (1926) studied fowl spirochetosis and made careful darkfield blood examinations. His findings were similar to those obtained by Meessen in cases suffering from Biermer's disease. It was difficult, at first, for Demnitz to determine whether these structures were artifacts or spirochetes. He also studied the blood of syphilitic rabbits by the darkfield, as well as the blood from the heart and various organs of chickens. The short forms of these "bodies," found by Demnitz, were morphologically strikingly similar to the "bodies" which Meessen thought were etiologically related to Biermer's disease. Demnitz had not yet determined (1926) whether these structures are present in healthy chickens. He does not believe that they have a specific etiologic significance.

Pelczar and Koloszynski (1931) report finding "motile" micro-thrombocytes in the blood of rats. They considered these bodies identical with Edelmann's kinetocytes. Edelmann (1931), however, emphatically disagrees with them. He states that the *kinetocytes* move actively in all directions and that these movements are not

"Brownschen Molekular-bewegungen," that they stain differently and are not to be confused with these micro-thrombocytes. He found that while the thrombocytes were greatly diminished, in two cases of thrombopenia, these kinetocytes were present as usual, and have nothing whatever to do with micro-thrombocytes as reported by Pelczar and Kolozynski.

In his report of two cases of chronic infectious anemia (March 5, 1925), Edelmann emphasized the presence of "outspoken eosinophilia," leading him to search for parasites in the blood. He found, in the red blood-corpuscles, and also extra-cellularly, "birnformige oder halbovale oder runde Gebilde," (also ring and three-pointed forms), which stained blue (protoplasm) and red (nucleus), with May-Giemsa stain. They appeared to Edelmann to be protozoa resembling "pirosomen," and thought they may prove to be the cause of infectious anemia.

Max Kahane, of Vienna (1895), discussed "Blastomyceten in Carcinomen und Sarkomen"; and in another paper he mentions fine "Sporen" in the blood of sarcoma patients.

A. P. Focker (1890), described "Hamatocyten" in his paper "Ueber eigenthumliche Evolutionsproducte des Hamoglobins."

Muller, of Nothnagel's Clinic, Vienna (July 1, 1896), in his paper "Ueber einen bisher nicht beachteten Formbestandtheil des Blutes" reviews the literature and discusses "die Zerfallsproducte von Leukocyten, etwa als frei gewordene Ehrlich Granula, ansprechen." He mentions Ehrlich's refractile "Korner," Kollicker's and Stohr's "Elementarkornchen" of fatty nature, Rollett's "fat droplets," Zimmermann's "Elementarkorperchen," "Kornchenbildungen," "Zerfallskorperchen," Hayem's "granulations libres," Schiefferdecker and Kossel's "fat droplets," "Fetttropfchen," "Kornchen," Kahane's "parasite," Bizzozero's "eiweissartigen Kornchen," etc. He calls the "bodies" he described "Blutstaubchen" or "Haemokonien."

Pelczar and Koloszynski (August, 1931), and Edelmann (June and August, 1931) overlooked the papers by H. Deetjen (1901 and 1909), and E. Frank (May 3 and 10, 1915) on blood-platelets and their "amoboide Bewegungen," and the statement by Georges Hayem (1878) on the changing forms of (blood-platelets) various blood elements.

Deetjen, H.: Untersuchungen uber die Blutplattchen, Virchows Archiv. f. Path. Anat. u. Physiol. 164: 239-263, 1901 (particularly pages 257-259).

Deetjen, H.: "Amoboide Bewegungen und Kern der Blutplattchen," Zeitschr. f. Physiol. Chem. 73: 1-26, 1909 (particularly pages 12-14).

Frank, E.: Essentielle Thrombopenie (Pseudohamophile), Berlin. klin. Woch. (May 3 and 10), 1915 (particularly page 491, second column, lines 11-67).

Hayem, Georges: Recherches sur l'evolution des hematies, Arch. de Physiol. 5: 707, 1878; and Du Sang, (Paris), p. 113, p. 389, and p. 859, 1889.

E. Frank (page 491), of the Medical Clinic of the University of Breslau, in his paper on "Essential Thrombopenia" (Pseudohemophilia) stated: "Trotz der Kernlosigkeit weissen die Plattchen eine Reihe von Lebensercheinungen auf: sie sind amoboider Bewegungen fahig (Deetjen), zeigen einen lebhaften respiratorischen Stoffwechsel (Morawitz und Lober) und enthalten peptid—spaltende Fermente (Aberhalden und Deetjen)."

Deetjen, H.: Handb. d. biochemischen Arbeitsmethoden (von Emil Aberhalden). 6: 383-388, 1912.

Schilling (Gradwohl's translation, 1929), speaks of parasitoid forms of "inner bodies," Ehrlich's hemoglobinemic inner bodies, Heinz's blue-granules, and Schmauch's inner bodies in cats. All these bodies are archoplasmatic and not karyogenic. He mentions nuclear spheres, "Jolly bodies," "Blutstaubchen" and "Haemokonien" or "hemokonia," *pseudospirochetes* (p. 274), and states that "upon heating, ameboid motion of all leucocytes takes places, with granular ciliation." In heated or severely anemic natural preparations (especially in fever), there occur short or long rodlets, spiral-shaped and with thickened poles, with peculiar indolent or passive motility, which develop from the "centers" of the erythrocytes; they are not present in a good smear. Typical spirochetes may be also simulated by chromatin threads, cholesterin fragments of erythrocytes and remnants of blood platelets. True spirochetes are always very fine and smooth.

Unstained Smear: *Darkfield*: Visible: (Schilling, p. 38): Erythrocytes, their rouleaux for-

mation and morphologic changes: blood platelets; lymphocytes as almost homogeneous globules with dark nuclear spaces; granulocytes (granulated: neutrophiles fine, eosinophiles coarse, somewhat yellowish); nucleus appears as a vacuole, also central apparatus; monocytes, fine dusty protoplasm. Upon heating, ameboid motion of all leucocytes (lymphocytes and monocytes very sluggish) takes place, with granular ciliation. After 3 to 10 minutes we see fibrin needles and formation, with motile debris and derivatives. The very small "powdery" particles of blood ("hemokonia") which become immediately visible under darkening or in the darkfield, are mainly fat particles. They are very much increased after meals. It is claimed that they constitute a clinical control of fat resorption and working capacity of the reticuloendothelium. Lack of hemokonia 1 to 2 hours after a fatty meal (butter, milk) indicates disturbed function of gall bladder or pancreas; their prolonged presence, a pathologic retention in the reticulum. The larger granules are "centers," etc.

Grawitz (Klin. Patholog. des Blutes, Leipzig, 1906, p. 205 and p. 210) discusses "Die Blutplattchen" and "Hamokonien."

Bizzozero speaks "Ueber die Blutplattchen" in Internationale Beitrage zur wissenshaftlichen Medizin I, 1891; and Virchows Archiv. Vol. 90. It is possible, too, that the peculiar "microorganisms" or "parasites" and the "motile blood-elements" reported by Edelmann in 1925, 1927, January 9, 1931, June 19, 1931, and August 28, 1931, in the study of cases of chronic infectious anemia, acute myeloblastic leukemia, and essential thrombopenia (pseudohemophilia or constitutional purpura) are perhaps being confused with Muller's (1896) "Blutstaubchen," or "Haemokonien," Deetjen's, Frank's and Pelczar and Koloszynski's motile "Blutplattchen" (thrombocytes).

Kahane's (1894 and 1895), Arnold's, Max Schultze's, van Niessen's (1895), Claus Schilling's (1925), Engel's (1893), Zeller's, Grassberger's, Siegel's, V. Schilling's, W. Kruse's, and Edwin Schultz's works must be carefully considered before reaching the conclusion that Edelmann's "kinetocytes" are "new hitherto unknown motile blood-elements." Also of interest in this connection are: Lilienfeld's studies, "Hamatologische Untersuchungen," in Arch. f. Anat. u. Physiol., 1892; Walther Kruse: Ueber Blutparasiten, Virchows Archiv., 120: 541-559, 1890 (Kruse mentions the "Korpern" found by O. Butschli and Gaule in frog's blood; on pages 543-545 he speaks of the "small actively motile Wurmchen und Korpern" in the blood); and Van Niessen: Ueber regenerative Vermehrung menschlicher Blutzellen, Virchows Archiv. 141: 252-264, 1895; (van Niessen noticed motile bodies in the blood).

November 12, 1931.

Primarius Dr. Adolf Edelmann, Direktor,
S. C. Childs' Spital and Institut,
IX, Pelikangasse, 15, Wien.

Dear Friend:

Since you and I began to study the peculiar "motile bodies" or "kinetocytes" (Edelmann), together, in Carlsbad, September 14, 1931, and during September and October on various occasions in the Childs' Institut in Vienna, I have carefully gone over the literature for the past sixty-five or seventy years.

It may be, perhaps, if we can find definite specific characteristics of our Edelmann kinetocytes, that positively differentiate them from all the other various "motile bodies" and "pseudoparasites" that have been previously described, and if we can demonstrate special qualities and special actions or functions of our newly-discovered motile element (?) and some differential response in cultural growth or staining, then we may hope that this is a new addition to hematology.

I wish to call your attention to many (motile) globular bodies, "pseudoparasites," "pseudospirochetes," etc., previously described by many authors.

May I suggest that you do *not* use *hot* paraffin to seal the cover-glass on the slide of the blood preparations? Febrile patients may show more changes in "motile bodies" (?) in the blood than afebrile. The important question to decide, is—are these "motile elements" or "kinetocytes" (Edelmann), the same that have previously been repeatedly described, or have we now a new motile body or "blood element"? Are these "Edelmann bodies" truly a new discovery? Are they anti-thrombin-like in action? Are they affected by fever, by heat, by chemical dyes, and

how? Are they the same or similar to those "moving bodies" seen by Beale (1864), Preyer (1864), M. Schultze (1865), Thoinot and Calmette (1892), Lewaschew (1892), Siegel (1905), Eve (1907), Porter (1907 and 1913), Balfour (1911), Seidelin (1912), Chambers (1913), Eberson (1920), E. W. Schultz (March, 1923), Zeller (1923), Determann (1925), Demnitz (1926), Morawitz (1926), Schilling (1926 and 1929), Grassberger (1928), Zadek and Burg (1930), and others?

Have Edelmann and Goldstein (beginning September 14th, 1931) been studying a genuine *new blood* element? Or are they only rediscovering an old thing? What are these motile elements?

I am enclosing three (3) pages containing about thirty references to the literature on this subject for your perusal and study.

With kindest personal regards to you, your good wife, Professor Silberstein, and your assistants, I remain

Sincerely and cordially yours,

HYMAN I. GOLDSTEIN, M. D.

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EDITORIAL

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W. EDWIN BIRD, M. D. ..... EDITOR
Du Pont Building, Wilmington, Del.

W. OSCAR LAMOTTE, M. D. ..... Associate Editor
Medical Arts Building, Wilmington, Del.

M. A. TARUMIANZ, M. D. ..... Associate Editor & Bus. Mgr.
Du Pont Building, Wilmington, Del.
Telephone, Wilmington, 4368

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THAT VETERANS' HOSPITAL

The Wilmington *Every Evening* for December 19, 1931, carried a front page story of the efforts being made in Washington to secure a veterans' hospital for this territory, to be located in or near Wilmington. The chief arguments seemed to be that veterans had available the services of only one physician, and only then for three hours a day, and that cases requiring hospitalization had to be shipped off to Philadelphia. If there were no hospitals in Wilmington there would be considerable justification for planning one, but the report, as given in the article cited, makes no mention of any investigation of the services, number of beds, costs, etc., that are already available here.

We wish to sound a word of caution: This whole government hospital plan bids fair to be the precursor of so-called "state medicine," which is repugnant to all physicians and to every thinking layman. The government has 40,000 beds for veterans, and proposes to build hospitals providing 100,000 beds more, at the trifling cost of $300,000,000, despite the fact that at the present time there are approximately 250,000 empty beds in the 8,000 civilian hospitals. For the government to persist in planning new beds, with an oversupply already on hand is no mere folly; it is a crime against the citizens.

That is not all: there is involved, should the plans proceed, a crime against the medical profession. With hospital plants totaling around 150,000 beds, there will be built up a personnel whose sole interest will be to hold on to their jobs by keeping all these beds filled. When the veterans are all dead, whom will these bureaucrats bring in to fill these 150,000 beds? Will the government donate these hospitals for civilian usage? Not if the jobholders can help it! One after another various groups of the civilian population will be granted hospitalization in government hospitals, at government expense, will be treated by government doctors working on salary, and will be withdrawn from the possible clientele of the already harassed private physician. The end of the story is perfectly obvious.

To head off this campaign requires education —of the profession, as to the jeopardy it is in; of the public, as to the uneconomic program proposed; of the legislators, as to their duty towards the *majority* of their constituents; of the veterans themselves, as to the vastly better services available in civilian hospitals, for the most part in their own home towns. Dr. Morris Fishbein recently addressed the House of Delegates of the Medical Society of Delaware on this very subject: read his remarks in THE JOURNAL for December, 1931, page 229. Then go talk to your veteran friends; they will see the light if it is put up to them properly; and finally, get in touch with our three representatives in Washington and give them the facts we feel sure they will thank you for.

AN ELASTIC CHARGE

We have often marvelled at the charge of "contempt of court." In law it is defined as "disobedience to, or open disrespect of, the rules or orders of a court, legislature, or the like." This strikes us as a very elastic definition, somewhat too broad, and definitely susceptible to various personal interpretations. Contempt is direct when it is committed in the presence of the judge or his officers (e. g., failure to answer a subpoena; refusal to testify, etc.), and is punishable immediately without a hearing, and, in most states, without any appeal. Contempt is indirect when it is not committed in the presence of the judge or his officers (e. g., tampering with a juror; intimidation of a witness; etc.), and is punishable only after a regular trial, and, in most states, with the right to appeal.

Scheffel, in his excellent *Medical Jurisprudence*, says that physicians, more than any other professional class, run a risk of committing contempt. The next most frequent group, according to our memory, must be editors and members of the press. Pity, then, the poor devil who happens to be both physician and editor; he certainly walks an uncertain path. Contempt by physicians, however, is usually unintentional, and the court, though speaking sternly, generally recognizes this fact: the physician who is actually sentenced for contempt is a rather rare species, fortunately.

Yet in the category of indirect contempt there is one classification that even the judges do not seem to have fully standardized in their minds, namely: criticism of the court, or criticism of the judge's judicial conduct. We dare say that among the judges will be found the same percentage of exaggerated ego that is to be found in all other educated classes; and that susceptibility to criticism varies proportionately. We dare say that legal acumen likewise varies, not only among judges but also among communities. We dare say that what constitutes contempt in one court or jurisdiction may not be contempt in another.

But criticism can be of all kinds, in diverse places, and at sundry times. The point to remember, if one *must* criticise a court, is to do so only *after* the particular trial or cause is ended. To criticise before or during a trial may conceivably, if the criticism receives sufficient currency or publication, obstruct justice, and therefore constitute contempt. But *after* a trial is over there is no judge in the whole United States whose official action is not subject to review or criticism, however unpalatable the latter may be. Judges, invariably referred to as "learned," are not demi-gods or supermen: they are simply successful lawyers who are honored with a great trust, but who, being human, have a definite percentage of error. Judges *have* been impeached, though this procedure is creditably rare; and judges *have* been reversed on appeal, though this is not particularly frequent. So, criticism *after* a trial is finally concluded is in order, provided it does not affect a pending appeal, or a case that is remanded for retrial, which means be sure the criticism is really *"after."* This version of the law on this subject was given wide publicity a few years ago in connection with a New York judge who cited and sentenced a city official who, *after* the trial was over, criticised the court. The official, on appeal, was found not guilty, and the country, through the press, was taught a very valuable bit of law.

With the above in mind, you may read the following press dispatch and come to your own conclusions:

EDITOR IN VIRGINIA JAILED
ON CONTEMPT CHARGE
(By The Associated Press)

HOPEWELL, Va., Dec. 31.—J. W. Mapoles, editor of the Hopewell News, wrote his editorials last night from a cell in the Hopewell jail.

He was sentenced yesterday to 30 days in jail for failure to produce the writer of a letter which was published in the open forum columns of the newspaper.

The letter which was signed "Jokester," said "If 16 efficient men of the Virginia bar, able and fluent, have no success in pleading a case, it looks like that many men might be 'hefty' enough to remove the court bodily."

It referred to Judge Thomas D. Robertson's recent action in fining the newspaper editor and the police chief $10 each because a police notation was published which the judge said insinuated he had freed a "big bootlegger." Sixteen attorneys appeared to represent the police chief.

Editor Mapoles published a letter, anonymously, *after* the trial; the letter referred to two fines for a notation also published, apparently, *after* the trial; the editor was not bound to violate a professional confidence *after* the trial, yet he languishes in jail! We face a dilemma: Is the New York decision wrong, or is Judge Robertson a candidate for impeachment? The vital thing is: just what *is* contempt of court?

EDITORIAL NOTES

DEAR DOCTOR:

THE JOURNAL and the Cooperative Medical Advertising Bureau of Chicago maintain a Service Department to answer inquiries from you about pharmaceuticals, surgical instruments and other manufactured products, such as soaps, clothing, automobiles, etc., which you may need in your home, office, sanitarium or hospital.

We invite and urge you to use this Service.

It is absolutely *free* to you.

The Cooperative Bureau is equipped with catalogues and price lists of manufacturers, and can supply you information by return mail.

Perhaps you want a certain kind of instrument which is not advertised in THE JOURNAL, and do not know where to secure it; or do not know where to obtain some automobile supplies you need. This Service Bureau will give you the information.

Whenever possible, the goods will be advertised in our pages, but if they are not, we urge you to ask THE JOURNAL about them, or write direct to the Cooperative Medical Advertising Bureau, 535 N. Dearborn St., Chicago, Illinois.

We want THE JOURNAL to serve *you*.

We take pleasure in calling your attention to the article by Dr. Goldstein, in this issue, on *Kinetocytes,* which represents, perhaps, a new and fourth cellular element of the circulating blood. This is the first article to appear in American literature on this subject, and while it is somewhat sketchy in character it is sufficiently complete to function as intended—a preliminary report. An excellent bibliography is included.

We take no pleasure, however, in clipping the following from the *J. A. M. A.* for December 26, 1931:

HEALTH AT WILMINGTON

Telegraphic reports to the U. S. Department of Commerce from eighty-two cities with a total population of 36 million, for the week ended December 12, indicate that the highest mortality rate (19.1) appears for Wilmington, and the rate for the group of cities, 11.3. The mortality rate for Wilmington for the corresponding period last year was 14.7, and the group of cities, 11.7. The annual rate for eighty-two cities for the fifty weeks of 1931 was 11.8, as against a rate of 11.9 for the corresponding weeks of 1930. Caution should be used in the interpretation of these weekly figures, as they fluctuate widely. The fact that some cities are hospital centers for large areas outside the city limits, or that they have a large negro population, may tend to increase the death rate.

The annual meeting of the New Castle County Medical Society was held in the Club Room of the Du Pont Biltmore Hotel on December 15, 1931. Sixty of President Niles' colleagues helped him make a success of that wild duck dinner he had promised us for seventeen years. At the business session the following were elected for 1932: President, Dr. George W. Vaughan; vice-president, Dr. Olin S. Allen; secretary, Dr. Douglas T. Davidson; treasurer, Dr. Norwood W. Voss; director, Dr. Albert J. Strikol; censor, Dr. Julian Adair. The resolution calling for an investigation into the conduct of the Office of Coroner was, by a vote of 36 to 24, carried over to the January meeting.

The inspector for the American College of Surgeons visited the four hospitals in Wilmington on December 18-19. We take it that all four will remain on their list of approved hospitals.

Tit for tat: now that they have inspected us, suppose we inspect them. What does the Martin Machine have to say about the following: (1) Maintenance of proper standards for admission to Fellowship; (2) Elimination of unworthy Fellows; (3) Publication of their financial statement?

Leave it to the old Kentucky home to lead the way. Their Woman's Auxiliary is now publishing a Quarterly, as a supplement to the *Kentucky Medical Journal.* The first issue of this publication, the first State Auxiliary publication in the country, appeared early in January. Physically it is a twin-sister to the state journals; editorially it is ably handled. The current issue contains twenty pages of text and reports, an excellent photograph of their president, eight pages of advertisements, and miscellany to make up a total of 32 pages. We congratulate the Kentucky Cardinalettes and wish them all success.

We call your attention to the yellow inserts, which began in the December issue. These European tours, under the direction of the Amerop Travel Service, are intended primarily, but not exclusively, for physicians and their families. If you cannot go yourself, remove the insert from THE JOURNAL and pass it on to some friend who may go. THE JOURNAL will receive a small commission from every passage booked from Delaware, so help your state publication by helping yourself and your friends to an unusually high-class trip to Europe this summer.

And, finally, to all our readers, wise and otherwise, THE JOURNAL extends its sincerest greetings for a Happy and Prosperous New Year.

DELAWARE PHARMACEUTICAL SOCIETY

Incompatibilities of Some Important Newer Chemicals*

By CHARLES F. LANWERMEYER
Chicago, Ill.

In his daily compounding of prescriptions, the pharmacist is often confronted with the problem of incompatibility. The physician knows what action he needs in the particular case and prescribes the individual drugs in combination; but it is up to the pharmacist to make an elegant, chemically and physically compatible mixture. The various textbooks on pharmacy devote some space to incompatibilities, and there are also books like Ruddiman's "Incompatibilities" devoted exclusively to this subject.

In this day of multifarious new chemical production, the authors of these texts would have to publish annual editions in order to keep step with the manufacturers. It was in order to fill this gap that the following work was undertaken.

This work presents the literature references in a condensed form, as well as the results obtained by the author in making mixtures such as the dispensing or manufacturing pharmacist might be asked to compound. The following chemicals, which are listed either in the U. S. P. or in the "New and Non-official Remedies," were used in this study: Acriflavine (Base), Acriflavine Hydrochloride, Amidopyrine, Barbital, Sodium Barbital, Benzocaine, Butesin, Butyn, Ephedrine Hydrochloride, Ephedrine Sulphate, Neonal, Phenobarbital and Procaine Hydrochloride. References to these items in the literature were examined, and combinations with other drugs were made up and allowed to age in the laboratory.

AMIDOPYRINE

(Also known as Pyramidon and Amidozone)

The solubility of Amidopyrine stated in the U. S. P. is 1 Gm. in 18 cc. of water. Other texts claim 1 in 11 (E) (X), 1 in 20 (Y), 1 in 9 (B). The author found that the U. S. P. solubility is correct. According to Comptes rend., 1927-185 p. 284, "the difference in water solubilities was accounted for because of this often being a mixture of A and B Pyramidon."

It is incompatible with acacia, producing a colored solution (R). This is due to the oxydase present, and can be prevented by heating the acacia to 85° C. which destroys the oxydase (S). A blue-violet color is produced by many oxidizing agents like ferric chloride, silver nitrate, nitric acid, spirit of nitrous ether, lead dioxide, iodine solutions and some enzymes (P). Mixed with aspirin even in two separate granulations (as in tablet manufacture) it will become yellow in a few months. If mixed and moistened in granulation it will become sticky and stick on the compressing machine (P). It is incompatible with amyl nitrite and apomorphine (BB). Mixed with citric or tartaric acids it forms a sticky mass in a short time, but with salicylic acid this occurs at once. With salol it becomes yellow colored in a short time. Dissolved in elixir lactated pepsin it changes the color of the elixir to purple and precipitates on standing. Dissolved in essence of pepsin it remains unchanged when fresh but precipitates on standing. With mercury bichloride it masses and becomes grey colored. With neonal it becomes sticky and develops a slight odor. Its aqueous solution has a slightly alkaline reaction. Mixed with the following there is no apparent change: antipyrine, acetphenetidin, ammonium chloride, borax, cinchophen, tannic acid, potassium acetate, sodium phosphate, sodium biphosphate, lead acetate, methenamine, menthol, phenolphthalein, potassium bromide, potassium iodide, sodium salicylate, extract of cascara and calomel.

ACRIFLAVINE (BASE)

Acriflavine is soluble in three parts of water (R), incompletely soluble in alcohol (T), nearly insoluble in ether, chloroform, liquid petrolatum and fixed and volatile oils (R). Its solutions have a neutral reaction (P), hydrochloric acid destroys its fluorescence (P). Solutions precipitate with silver nitrate (P), sodium hydroxide gives an orange precipitate in dilute solutions (P). Solutions 1 to 1000 can be made in normal saline solution (X). A solution in oil can be made by dissolving 2% (based on the final) in a small amount of alcohol and then diluting with castor oil q. s. (X).

ACRIFLAVINE HYDROCHLORIDE

(Also known as Trypaflavine)

Acriflavine hydrochloride is soluble in three parts of water (BB), although commercial sam-

* From the Pharmaceutical Research Department, Abbott Laboratories.

ples vary considerably (T). It is soluble in alcohol 1 in 40 (BB), insoluble in ether, chloroform, liquid petrolatum, fixed or volatile oils (T), insoluble in oleic acid and eucalyptol (BB). Its solutions have a distinct acid reaction making it incompatible with carbonates or bicarbonates (T). It is incompatible with Dakin's solution and other chlorine antiseptics, mercury bichloride and phenol in solution (BB). A solution in normal saline solution is clear when freshly prepared, but precipitates in 24 hours, although if the saline solution contain 5% salts, precipitation takes place at once (BB). It is compatible with an 0.5% sodium citrate solution. Its solution in water is stable even when boiled or autoclaved at 130° C. Stains can be removed with a little dilute hydrochloric or sulphurous acid, followed by washings with water.

ANESTHESIN

(Also known as Benzocaine, Ethylaminobenzoate or Keloform)

Heated with soluble alkali hydroxides and carbonates Anesthesin decomposes into alcohol and p-aminobenzoic acid (P). Aqueous solutions precipitate with iodine (P), small amounts of hydrochloric acid increase its solubility (P). It is incompatible with acids or acid salts in solution (W). Its aqueous solution is decomposed by boiling, but solutions in oil can be boiled (W). If used in combinations containing perfume materials like vanillin or heliotropin even in small amounts, discoloration will result. Mixed with bismuth subnitrate it discolors in a few months; with resorcin it forms a sticky mass and discolors on standing. Mixed with the following substances no apparent change takes place: bismuth subgallate, zinc sulphocarbolate, sodium bicarbonate, magnesium oxide, bismuth subsalicylate, salol, sodium thiosulphate, acetanilid, boric acid, tincture ferric chloride, milk of bismuth, milk of magnesia, calomel, cerium oxalate and ammonium bromide.

BARBITAL

(Also known as Veronal, Barbitone)
(Hypnogen or Malonal)

Barbital and acetphenetidin are antagonistic physiologically, as tested on guinea pigs (Arch. Expt. Pharm. & Pharmacol., 116-140-6 (1926). Antipyrine is antagonistic to the sedative effect of barbital, but the stimulating effect of the two is synergistic (Arch. Expt. Pharm. & Pharmacol., 114-313-26 (1926). Heating with alkali carbonates or hydroxides decomposes it, liberating ammonia (P). It darkens with calomel slowly; but when moist or if sodium bicarbonate is present it darkens immediately (P). With alkalies it forms salts that are soluble in water (P).

BARBITAL SODIUM

(Also known as Medinal)

Barbital sodium is incompatible with ammonium salts (viz., ammonium bromide) giving off ammonia and precipitating barbital; (barbital itself is not changed in this way (H). It is incompatible with acid salts, morphine hydrochloride and with solutions containing fruit essences on account of the acid present (H). With tincture of cinchona it precipitates out the alkaloids, but the reaction takes 24 hours.

BUTESIN

(Also known as Scuroforme)

Butesin is almost insoluble in water, and when boiled in it is slowly hydrolyzed; it is soluble in dilute acids, alcohol, chloroform, ether, benzene and fatty oils. Its solution in oil is not decomposed by heat. It is incompatible with silver nitrate (R). It dissolves very readily in oils of cassia, cloves and wintergreen; with resorcin it masses at once and changes to a green sticky mass. It is insoluble in liquid petrolatum. With the following in dry mixtures there is no apparent change: bismuth subnitrate, calomel, betanaphthol and sulphur; also sulphur ointment.

BUTYN

Butyn is incompatible with alkali carbonates and hydroxides, precipitating out the free base, with soluble bicarbonates precipitating out the carbonate (R); it precipitates with the general alkaloidal reagents (P); it is incompatible with iodine solution, barium chloride and picric acid (R). It decolorizes potassium permanganate solution. It is incompatible with chlorides or saline solutions, but isotonic solutions can be made using potassium sulphate in the following amounts:

½% solution requires 1.34 Gm. K_2SO_4 per 100 cc.

1% solution requires 1.21 Gm. K_2SO_4 per 100 cc.

2% solution requires 0.97 Gm. K_2SO_4 per 100 cc.

5% solution requires 0.24 Gm. K_2SO_4 per 100 cc.

It is incompatible with silver protein preparations in solution, precipitating yellowish brown at once. With lead acetate solution it precipitates lead sulphate at once, but makes a good shake mixture. With fluid extract of hydrastis, fluid golden seal compound, and solution hydrastine there is no apparent change.

EPHEDRINE HYDROCHLORIDE

Ephedrine hydrochloride is soluble in water 1 to 7, in alcohol 1 to 8, the solutions being neutral. It is insoluble in ether (BB) (T). It is incompatible with silver salts, causing precipitation, with amidopyrine in solution it precipitates; and it is incompatible with syrup of wild cherry. With the following there is apparently no change: elixir euphorbia compound, potassium bromide, potassium iodide, aspirin, amidopyrine (dry), acetanilid, acetphenetidin, barbital, phenobarbital, neonal, calomel, sodium bicarbonate, magnesium carbonate and oxide, phenolphthalein, quinine sulphate, sodium salicylate, neocinchophen, methenemine, potassium acetate, caffein, caffein citrated, elixir terpin hydrate, brown mixture, and syrup of white pine compound.

EPHEDRINE SULPHATE

Ephedrine sulphate has practically the same properties as the hydrochloride, except that it is less soluble in cold alcohol, is compatible with silver salts and is incompatible with potassium iodide causing yellow discoloration of the solution, forming a precipitate; with neonal it causes a strong etheral odor, with methenemine a strong urine-like odor; with potassium acetate a strong odor is also developed. With all the other combinations mentioned above under ephedrine hydrochloride there was no apparent change.

NEONAL

Neonal is incompatible with acetanilid, developing a foreign odor. A mixture with amidopyrine becomes sticky and develops an odor. With calomel, especially if any moisture is present, the mixture turns grey although if perfectly dry there is apparently no change. With salicylic acid it develops an odor. With the following there is apparently no change: acetphenetidin, ammonium bromide, bismuth subnitrate, magnesium oxide, sodium bicarbonate, sodium bromide and sodium salicylate.

PHENOBARBITAL SODIUM

Phenobarbital sodium is unstable unless stored in airtight containers in the dark (H). In aqueous solution, on standing or by boiling, it is partly decomposed with formation of phenyl ethyl acetyl urea, which precipitates out (Y). On account of its alkaline reaction it has the same incompatibilities as the alkalies, and may therefore precipitate alkaloids out of solutions of their salts such as caffeine and strychnine out of elixirs and syrups (L). It is incompatible with ammonium bromide in solution, precipitating out the phenobarbital (H), the supernatant liquid having an ammonia odor (L).

PROCAINE HYDROCHLORIDE
(Also known as Ethocaine, Kerocaine)
(Neocaine, Novocaine and Syncain)

Novocaine is incompatible with iodine and with picric acid solution (D). Also with mercuric chloride, gold chloride and Mayer's reagent (P), and with silver salts (R). With sodium carbonate or hydroxide solutions, the base separates out as an oily liquid (D) (P). With sodium bicarbonate there is no change (P). It is incompatible with tannin, calomel, potassium dichromate and permanganate (BB). Its solutions can be sterilized at 100° C. without change, but are damaged at 120° C.

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MISCELLANEOUS

The Genesis of Social Insurance

EDWARD H. OCHSNER, M. D.
Chicago, Ill.

Social insurance is the hybrid offspring of impracticable sentimentalism and political expediency. It is an epidemic disease first observed in Germany about fifty years ago which has gradually spread and infected a considerable number of the nations of the earth and now has arrived at our very doors. Unless we succeed in establishing a rigorous quarantine of enlightened public opinion, it will surely gain a foothold in this country in the not distant future.

Social insurance consists of the following subdivisions or parts: Compulsory health insurance, old age pensions, widows' and orphans' pensions, and unemployment pensions or doles. In none of the countries were they all adopted at the same time. Germany adopted compulsory health insurance in 1883, and all of the other forms since that time. Austria adopted compulsory health insurance in 1888; Hungary, in 1891. England adopted old age pensions first and compulsory health insurance in 1911, and the others subsequently. In this country some of the states have adopted old age pensions and some widows' and orphans' pensions, but so far none have adopted compulsory health insurance, for which negative blessing let us raise our voices in thanksgiving.

When the scientific physician is confronted with the problems presented by a new patient, he meets the situation in the following manner: he obtains a complete family and personal history in order to ascertain if possible the causes which have brought about the condition; by his physical examination and laboratory investigations he finds out what variations from the normal have taken place; after all this he is in a position to advise and institute the proper treatment. Let us follow the same course in the study of this problem.

During the late seventies a number of German parlor socialists conceived the idea that the state make itself responsible for the medical care of its workers. The sentiment in favor of compulsory health insurance grew rapidly among the workers, and Bismarck, although expressing serious doubts as to the soundness of such a measure, yet feeling that something had to be done in order to appease the clamor of the proletariat and the alarming growth of socialism, adopted social insurance as a government measure, had a bill drafted and enacted into law.

In England, National Insurance, as it is called there, had a slightly different setting, but substantially the same background. In 1910, David Lloyd George, in order to strengthen himself politically, decided the time for such legislation was opportune. Not being able to speak German, he gathered about himself several interpreters, hied himself to Germany and after interviewing the well-paid heads of the German system, and after having been wined and dined and lionized for two weeks or so, he returned to England very enthusiastic about the whole project, had a law drafted, and later secured its passage. In the recent parliamentary election the Liberty party, of which Lloyd George has been the head for many years, elected just four members to Parliament or less than one per cent of the whole number. So while Lloyd George may have saved his political skin by National Insurance in 1911, he certainly lost his hide by it in 1931.

Practically every reform movement attracts to itself a considerable number of well-meaning, emotionally impressionable, impractical, irresponsible, very vociferous individuals, and very often a group, usually the very ones who manage the propaganda and who hope to gain some pecuniary benefit from it. Social Insurance is no exception to this general rule.

Some of the common characteristics of reformers is that they want a new law passed for every human ill, and when the law is enacted, they either sit back waiting for the millenium to arrive or they rush off looking for new evils to correct by new laws, forgetting to see to it that the law just passed is being properly enforced, and forgetting at all times that all laws must depend for their enforcement not upon supermen, but upon men often of less than average intelligence and integrity, upon politicians and their henchmen, who are quick to see how these usually unsound and loosely drawn laws can be converted to their own advantage. The above is what is actually happening in some of the countries where such laws are in operation, as future installments will show.

Co-operation in Necropsy Work

The percentage of necropsies performed in this country is still lamentably low. A properly performed necropsy is not only necessary in many cases to establish the exact cause of death, but is one of the chief means of advancement of medical knowledge. The reasons for failure to obtain necropsies are obvious. The physician in charge of a case lacks the scientific urge, the relatives of the deceased are opposed, and the mortician is, to say the least, cool, because necropsies may disturb the routine of his work. A joint committee of the New York Academy of Medicine, the New York Pathological Society and the Metropolitan Funeral Directors' Association recently held a series of meetings to discuss the problem. A substantial agreement was reached. All agreed that a necropsy by a pathologist is desirable in order to advance knowledge concerning the nature of the disease and to provide reliable records. The performance of necropsies in a hospital is a constant influence to improve the service and to help physicians serve the next patient with greater confidence and skill.

The hospital and its staff have not completed their services to the family on the death of the patient, but they owe to the family an account of what has occurred. This requires that a representative member of the family come to the hospital for a personal interview and to give permission for examination of the body of the deceased. The funeral director must recognize this relationship and should not oppose the discharge of this obligation. Unreasonable delay by the hospital in its attempt to obtain permission for necropsy is objectionable to a funeral director, as it may require him to cancel contracts for transportation and other services and thus increase his expense. The conflict of interests requires mutual consideration and co-operation. Arrangements should be worked out in every hospital whereby the unnecessary loss of time on the part of the funeral director may be obviated. Hospital employees in general must not give information to funeral directors or to any other unauthorized person in regard to persons critically ill or dead in the hospital. The hospital authorities should be certain that the necessary data for a death certificate are on the chart at the time of admission of the patient, with exception of the facts relating to the nature, progress and termination of the disease. Any interference by a funeral director with the legitimate efforts of the hospital to obtain permission for necropsy shall be regarded as a reportable grievance. The necropsy may be performed in a way to facilitate the work of the embalmer. The main longitudinal incision, the committee believes, should end over the midsternum, and curved incisions with the concavity toward the head should be carried laterally to each shoulder. When the skull is opened, the frontal bone should be cut above the hairline and the base of the skull cemented with plaster before the skull cap is replaced. When the neck organs and tongue are removed, the head should be embalmed through the carotids unless there is a serious contra-indication. Other recommendations made by the committee as to technic are that necropsies should be completed at the time promised, if there has been an advance agreement, and that a representative of the funeral director should be a welcome associate at the necropsy table. The spirit of co-operation shown in the report of the joint committee is commendable. Similar co-operation everywhere would no doubt result promptly in an increase in the percentage of necropsies.

Gradually, competent embalmers are tending toward the encouragement of postmortem examination. In the November issue of the *Casket and Sunnyside,* a publication for morticians, appears an editorial commenting on a previous statement on this subject by THE JOURNAL. The concluding statement of this editorial reads:

> We might as well recognize, first as last, that the welfare of the living, the public health, is the chief concern of the public, and that to permit the medical profession to secure sufficient scientific data, autopsies must be performed. We might as well also decide that if we explain our difficulties to the medical practitioners, rather than antagonizing them, they will be willing to co-operate with us in avoiding unnecessary mutilation of bodies committed to them for autopsy. Finally, we might as well realize that if we say "an autopsied body can't be embalmed or so prepared that family and friends may view it," we're stultifying ourselves and our claims to professional standing, and admitting our own incompetence.

The editorial points out further that the mortician should be proud of his ability to make presentable a body that has been subjected to postmortem examination and that the cry against such examinations on the part of morticians must be taken as a confession of incompetence. Such

support from one of the leading trade publications in the field is an encouraging sign to scientific medicine. It means that the pathologist will now need only to instruct the family and not to convince the mortician. — Editorial, *Jour. A. M. A.*, Dec. 19, 1931.

Present Status of Anesthesia Problem

ARTHUR DEAN BEVAN, Chicago (*Journal A. M. A.*, Nov. 21, 1931), reviews briefly the subject of anesthesia, tests the various anesthetic agents by the scheme of measurement which his associates and he have devised, and attempts in a judicial way to determine what anesthetics are today the safest, most efficient and most practical for use in a general surgical clinic. He believes that the use of chloroform, of intraspinal anesthesia, of intravenous anesthesia, of intrarectal anesthesia, of intratracheal anesthesia and of the so-called basic anesthetics, such as scopolamine, avertine and amytal must be limited to very narrow fields. Fortunately, local anesthesia, gas anesthesia and ether afford three anesthetic measures which, if handled by an expert can be used alone or in sequence, with abolition of pain and, if desired, the abolition of consciousness and, when required, complete relaxation, and can secure complete and safe anesthesia for any and all surgical operations. This places anesthesia on a very unpretentious, simple basis, but here, as in all fields of surgery, it finally becomes apparent that simplicity is near truth. The author believes that the general adoption of this simple scheme of anesthesia will prevent many anesthetic accidents and save many lives.

Management of Skull Fractures and Intracranial Injuries

HARRY E. MOCK, Chicago (*Journal A. M. A.*, Nov. 14, 1931), calls attention to the fact that the annually increasing morbidity and mortality rate due to trauma, with skull fracture causing a high percentage of the deaths, makes this one of the great economic and medical problems of the present time. It is impossible to standardize treatment, as each individual case presents its own peculiar requirements. But it is possible to give a rational routine treatment which can be applied to 50 per cent of all skull fracture cases and then to classify the remaining cases into the following three groups: (1) Those in which rest treatment alone is sufficient (4 per cent); (2) those who must have, in addition to routine care, the special treatment of lumbar drainage (33 per cent), and (3) those cases having definite, recognized indications for cerebrocranial operations (13 per cent). The author attempts to clear up certain controversies by detailing those practices recognized by the majority of authors on this subject and proved of the greatest value in his hands, in the management of skull fractures and cerebrocranial injuries. His article was written for those men away from medical centers who are just as frequently confronted with these cases and who are sometimes led astray in their treatment by certain teachers decrying lumbar drainage and by other teachers extremely adept in operative technic, advocating operative intervention in skull fractures, especially decompressions. He believes that if the average man will delay all roentgen examinations, undue physical examinations and operative procedures (with a rare exception) until the initial shock is over and then will classify his cases according to their signs and symptoms, he will develop for himself a common sense, rational line of treatment free from many of the controversial pitfalls commonly found in the management of skull fractures. Skull fractures should be treated at or near where they occur. Specialists, if desired, should be taken to the patient with the skull fracture rather than the patient to the specialist. Since, in the majority of communities, specialists in this condition do not exist and since the automobile has become a potential carrier of skull fractures to every hamlet, village and city in the land, it behooves all with experience in this matter to simplify and clarify the management of skull fractures to the end that the majority of physicians can properly cope with this grave emergency when and wherever confronted with it.

Prevention of Experimental Duodenal Ulcer by Feeding Neutral Gastric Mucin

M. S. KIM and A. C. IVY, Chicago (*Journal A. M. A.*, Nov. 21, 1931), observed that duodenal ulcers occurred spontaneously in six of ten dogs with biliary fistula. In seventeen biliary fistula dogs (about 25 pounds in weight) receiving 15 Gm. of neutral gastric mucin twice daily with their food, no ulcers developed. On the basis of these observations, the authors conclude that the administration of mucin prevents ulcer formation in dogs with biliary fistula.

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